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BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Application Number: 09/558,472
Filing Date: April 25, 2000
Appellant(s): BRISTOW ET AL.

Steven L. Highlander
For Appellant

EXAMINER'S ANSWER

This is in response to the appeal brief filed 11-1-06 appealing from the Final Office action mailed 3-19-04 and the Advisory action mailed on 7-9-04.

(1) Real Party in Interest

A statement identifying by name the real party in interest is contained in the brief.

(2) Related Appeals and Interferences

The examiner is not aware of any related appeals, interferences, or judicial proceedings which will directly affect or be directly affected by or have a bearing on the Board's decision in the pending appeal.

(3) Status of Claims

The statement of the status of claims contained in the brief is correct.

(4) Status of Amendments After Final

The appellant's statement of the status of amendments after final rejection contained in the brief is correct.

(5) Summary of Claimed Subject Matter

The summary of claimed subject matter contained in the brief is correct.

(6) Grounds of Rejection to be Reviewed on Appeal

The appellant's statement of the grounds of rejection to be reviewed on appeal is correct.

(7) Claims Appendix

The copy of the appealed claims contained in the Appendix to the brief is correct.

(8) Evidence Relied Upon

No evidence is relied upon by the examiner in the rejection of the claims under appeal.

(9) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

Claim 23 is rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The claim is directed to a method of treating myocardial failure in a human comprising administering an effective amount of a transgene encoding for alpha-myosin heavy chain (MHC), wherein expression of alpha-MHC provides improvement in left ventricular ejection fraction.

The specification discloses a method of myocardial gene therapy to increase alpha-MHC expression by delivering a transgene encoding alpha-MHC to a human so that the alpha-MHC transgene is expressed in the myocardial tissue of the heart (e.g. p. 14, lines 20-28). The specification further discusses construction of the transgene (e.g. p. 15) and modes of delivery of the transgene (e.g. p. 16, lines 4-15). The specification shows that failing human left ventricle exhibited a significant reduction in alpha-MHC as compared to non-failing controls by RT-PCR analysis (e.g. p. 35, lines 2-4), and up-regulation of alpha-MHC mRNA in myocardial tissue in human subjects suffering from idiopathic dilated cardiomyopathy, who received medical treatment with beta-blocking agent carvedilol or metoprolol (e.g. Example 5).

The claim encompasses administering a vector comprising a transgene or a naked transgene encoding for alpha-MHC to a human for treating myocardial failure via various administration routes so as to provide improvement in left ventricle ejection fraction. The claim

reads on gene therapy in vivo. The nature of the invention being gene therapy, the state of the prior art was not well developed and is highly unpredictable. Eck et al., 1996 (Goodman & Gilman's The Pharmacological Basis of Therapeutics, McGraw-Hill, New York, p. 77-101) reports that numerous factors complicate in vivo gene therapy with respect to predictably achieving levels and duration of gene expression which have not been shown to be overcome by routine experimentation. These include, the fate of the DNA vector itself (volume of distribution, rate of clearance into the tissues, etc.), the *in vivo* consequences of altered gene expression and protein function, the fraction of vector taken up by the target cell population, the trafficking of the genetic material within cellular organelles, the rate of degradation of the DNA, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, and the protein's compartmentalization within the cell, or its secretory fate, once produced. These factors differ dramatically based on the vector used, the protein being produced, and the disease being treated (e,g, bridging pages 81-82). The specification fails to provide adequate guidance for how to overcome any of the above unpredictable parameters in the gene therapy art such that one would be able to achieve therapeutic alpha-MHC transgene expression in target cells in a human subject with myocardial failure. It is important to note that treatment encompass complete amelioration of symptoms associated with myocardial failure or cure of myocardial failure. The specification fails to provide adequate guidance for how the administration of a transgene encoding alpha-MHC and mere expression of said transgene would be sufficient for treating myocardial failure in a human.

Further, vector targeting to desired tissues in vivo continues to be unpredictable and inefficient as supported by numerous teachings in the art. For example, Miller (1995, FASEB J.

Vol. 9, p. 190-199) reviews the types of vectors available for in vivo gene therapy. And conclude that "for the long-term success as well as the widespread applicability of human gene therapy, there will have to be advances...targeting strategies outlined in the review, which are currently only at the experimental level, will have to be translated into components of safe and highly efficient delivery systems" (e.g. p. 198, column 1). Deonarain (1998, Expert Opin. Ther. Pat., Vol. 8, pages 53-69) indicates that one of the biggest problems hampering successful gene therapy is the "ability to target a gene to a significant population of cells and express it at adequate levels for a long enough period of time" (page 53, first paragraph). Deonarain reviews new techniques under experimentation in the art which show promise but states that such techniques are even less efficient than viral gene delivery (see page 65, first paragraph under Conclusion section). Verma (Sept. 1997, Nature, Vol. 389, pages 239-242) reviews vectors known in the art for use in gene therapy and discusses problems associated with each type of vector. The teachings of Verma indicate a resolution to vector targeting has not been achieved in the art (see entire article). Verma also teaches appropriate regulatory elements may improve expression, but it is unpredictable what tissues such regulatory elements target (page 240, sentence bridging columns 2 and 3). Crystal (1995, Science, Vol. 270, page 404-410) also reviews various vectors known in the art and points out that "among the design hurdles for all vectors are the need to increase the efficiency of gene transfer, to increase target specificity and to enable the transferred gene to be regulated" (page 409).

In addition, Nabel (Circulation, 1995, pp. 1-17) teaches that the goal of development of cardiovascular gene therapy depend on technical advances in the development of methods of gene delivery, long-term, highly-efficient and targeted expression to relevant cells, and vectors

that are safe for human administration (e.g. p. 1, bottom of page, p. 2, top of page). Nabel further teaches that myocardial gene therapy has been hindered by limited transfection efficiency, transient expression of recombinant genes, and vectors that have provoked inflammatory responses (e.g. p. 5-6, Myocyte Gene Transfer). Hajjar (Circulation Research, 2000, pp. 1-12) teaches that "bridging the gap between [these] basic investigative studies and clinical gene therapy remains a formidable task" (e.g. p. 1, abstract, lines 10-11). Hajjar further teaches that relatively few vectors exist which achieve high-level transgene expression in post-mitotic cells, such as cardiomyocytes, and that these vector evoke a robust immune response (e.g. p. 2, 2nd paragraph, lines 4-7). Because of this immune response, Hijjar states that clinical applications will require other vectors or further refined vectors (e.g. p. 3, 1st paragraph, lines 5-7). Hajjar further teaches that "it is important to acknowledge that the field of gene therapy has not yet proven its clinical value in any context" (e.g. p. 2, 3rd line from the bottom) and that "Optimizing conditions for gene transfer into large animals and eventually humans will require substantial further investigation" (e.g. p. 4, 1st paragraph, last sentence).

Furthermore, the specification fails to provide a correlation to therapeutic levels of expression of alpha-MHC transgene in vivo in any subject having myocardial failure. An increase in the amount of alpha-MHC mRNA in myocardial tissue does not provide a prediction of therapy for any subject having myocardial failure because of those factors that complicate the unpredictability of gene therapy in vivo as discussed above, for example, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, the protein's compartmentalization within the cell, or its secretory fate, once produced, and the biological function of the protein. The specification fails to provide adequate guidance and

evidence for a correlation between therapeutic levels of expression of alpha-MHC and improvement in the left ventricular ejection fraction in vivo.

In view of the unpredictability of in vivo gene therapy in general, and particularly, unpredictability in myocardial gene therapy in vivo, and the lack of a correlation between therapeutic levels of expression of alpha-MHC and treatment of myocardial failure, such as improvement in the left ventricular ejection fraction in vivo, one skilled in the art at the time of the invention would not know how to treat myocardial failure in a human by administering a transgene encoding alpha-MHC such that expression of alpha-MHC would be able to provide therapeutic effect for treating said myocardial failure, such as improvement in left ventricular ejection fraction.

For the reasons discussed above, it would have required undue experimentation for one skilled in the art at the time of the invention to practice over the full scope of the invention claimed. This is particularly true given the nature of the invention, the state of the prior art, the breadth of the claims, the level of one of ordinary skill which is high, the amount of experimentation necessary, the lack of working example and scarcity of guidance in the specification, and the unpredictable nature of the art.

(10) Response to Argument

Appellants cite James et al., 2004 (Abstract presented at the Keystone Meeting on "Biology of Cardiac Disease") and argue that addition of alpha-MHC transcripts via a transgene strengthens the heart and renders it resistant to tachycardia-induced cardiomyopathy. The rabbit heart is constituted much like the human heart. The abstract shows that adding as little as 10-15% more alpha-MHC than baseline is protective to the rabbit heart, which can be extrapolated

into human heart. Appellants further argue that whether or not the gene is present in every cell in the body is completely irrelevant, and the gene used in the Abstract by James is the same as the gene used in the claimed method and it is expressed in a measurable amount that provides guidance on a therapeutic level to clinicians (Brief, p. 5). This is not found persuasive because of the reasons of record and the following reasons.

Firstly, the Abstract by James teaches preparation of transgenic rabbit expressing varying proportions of ventricular alpha-MHC replacement ranging from 10-15% to about 50% and shows that persistent expression of alpha-MHC in said transgenic rabbit is protective during tachycardia induced cardiomyopathy (TIC). Although both the gene used in the Abstract and the instant invention are alpha-MHC, however, constantly expressing alpha-MHC at 10-15% to about 50% in a transgenic rabbit to be protective during TIC, as taught in the Abstract by James, is totally different from the claimed method of the instant invention, i.e. administering a transgene encoding alpha-MHC to a human via various administration routes so as to provide therapeutic effect for treating myocardial failure, such as improvement in left ventricular ejection fraction. In the transgenic rabbit, the alpha-MHC gene into the genome of every cell in said rabbit and about 10-15% to about 50% of alpha-MHC is expressed in the heart cells. However, the instant invention requires administration of a transgene encoding alpha-MHC to a human via various administration routes and it is unclear whether sufficient transgene can reach the target cells, i.e. heart cells, and whether sufficient alpha-MHC protein can be expressed and obtained in sufficient duration of time so as to provide therapeutic effect for treating myocardial failure, such as improvement in left ventricular ejection fraction. Therefore, this post-filing Abstract by

James does not teach the same method as claimed in the instant invention and the evidence provided in the Abstract fails to provide enabling disclosure for the claimed invention.

Secondly, indeed, whether or not the gene is expressed in every cell in the body is irrelevant, however, the instant invention requires expression of the administered alpha-MHC transgene at the target cells, i.e. heart cells, in vivo which was unpredictable at the time of the invention for the reasons of record and as discussed above under the ground of rejection section. Numerous factors complicate in vivo gene therapy with respect to predictably achieving levels and duration of gene expression which have not been shown to be overcome by routine experimentation. These include, the fate of the DNA vector itself (volume of distribution, rate of clearance into the tissues, etc.), the *in vivo* consequences of altered gene expression and protein function, the fraction of vector taken up by the target cell population, the trafficking of the genetic material within cellular organelles, the rate of degradation of the DNA, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, and the protein's compartmentalization within the cell, or its secretory fate, once produced. These factors differ dramatically based on the vector used, the protein being produced, and the disease being treated. Vector targeting to desired tissues in vivo continues to be unpredictable and inefficient as supported by various teachings in the art. Specifically, myocardial gene therapy has been hindered by limited transfection efficiency, transient expression of recombinant genes, and vectors that have provoked inflammatory responses. Relatively few vectors exist which achieve high-level transgene expression in post-mitotic cells, such as cardiomyocytes, and that these vector evoke a robust immune response. Thus, it was unpredictable whether sufficient alpha-MHC transgene can reach target cells via various

administration routes, and sufficient alpha-MHC protein can be expressed and obtained in sufficient duration of time so as to provide therapeutic effect for treating myocardial failure, such as improvement in left ventricular ejection fraction.

Thirdly, the specification fails to provide a correlation to therapeutic levels of expression of alpha-MHC transgene in vivo in any subject having myocardial failure. An increase in the amount of alpha-MHC mRNA in myocardial tissue does not provide a prediction of therapy for any subject having myocardial failure. The specification fails to provide adequate guidance and evidence for a correlation between therapeutic levels of expression of alpha-MHC and symptoms of myocardial failure.

Appellants cite a variety of reference regarding gene therapy cited in previous response, including Alexander et al., 1999, Chien et al., 2000, Davidson et al., 2001, Pachucki et al., 2001, Shinmura et al., 2000, Silva et al., 2000, Lenhart et al., 2000, Lazarous et al., 1999, and Wickenden et al., 1999, and argue that those references teach successful transfer of genes into cardiac tissue (brief, p. 6). This is not found persuasive because of the reasons of record and the following reasons. The cited references Alexander, Chien, Davidson, Shinmura, Lenhart, and Lazarous teach direct injection or ex vivo perfusion of vector expressing reporter gene LacZ, GFP, or human beta2-adrenergic receptor to cardiac tissue. References Pachucki and Wickenden teach preparation of transgenic mice expressing human D2 under the control of alpha-MHC promoter and expressing a dominant-negative N-terminal fragment of the Kv4.2 pore-forming potassium channel subunit under the control of the mouse alpha-myosin heavy chain promoter, respectively. Reference Silva teaches generation of transgenic rat expressing the human tissue kallikrein gene. None of the cited references teach the claimed method of the instant invention

and the reporter genes and other genes used in the cited reference are not the alpha-MHC gene as claimed. Since the unpredictable nature of the in vivo gene therapy in general and specifically the in vivo myocardial gene therapy as discussed above, each in vivo gene therapy has to be considered individually. The cited reference Eck refers to the unpredictable nature of all in vivo gene therapy in general (see page 78-81, Table 5-1), therefore, the teachings of using reporter genes or other therapeutic genes for in vivo gene therapy by the cited references of appellants do not solve the unpredictable nature of the in vivo gene therapy in general or specifically the in vivo myocardial gene therapy. One successful in vivo gene therapy cannot be extrapolated into success for another gene therapy in vivo. Further, the genes used in the cited references are different from the alpha-MHC gene used in the instant invention. It was well known in the art that different proteins have different biological functions. As discussed above, numerous factors complicate in vivo gene therapy with respect to predictably achieving levels and duration of gene expression. These factors include the gene used, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, the biological function of the protein, and the disease being treated. Therefore, whether administration of a transgene encoding alpha-MHC to a human would be able to provide therapeutic effect for treating myocardial failure in vivo would require specific guidance and evidence, which is absent in the instant invention. Thus, the cited references could not constitute a success of the myocardial gene therapy in vivo as claimed.

Appellants cite references Yue, Schroeder, O'Donnel, del Monte, and Fromes, and argue that examiner failed to convincingly rebut the claims by those references. Appellants specifically states that Fromes describe the successful delivery of a gene to the myocardium by

intraperitoneal injection and states that "gene therapy is a potential new strategy for cardiovascular diseases" (brief, p. p. 6-7). This is not found persuasive because of the reasons of record and the reasons set forth above in the preceding paragraph. Yue teaches AAV-mediated gene transfer expressing microcystrophin to the newborn mdx mouse cardiac cavity (e.g. Methods and Results, p. 1). Schroder teaches injecting recombinant adenovirus expressing beta-gal into syngeneic rat heart transplants via the proximal aorta (e.g. p. 191, left column). O'Donnell teaches in vitro adenovirus-mediated gene transfer of SERCA1 gene or GFP gene under the control of CMV promoter (e.g. abstract). Del Monte teaches adenovirus-mediated gene transfer of SERCA2a gene or GFP gene via catheter-based technique in a rat model (e.g. Methods and Results, p. 1424). Fromes teaches adenovirus-mediated gene transfer of beta-gal via injection into the pericardial sac of adult rat (e.g. abstract). It should be noted that Fromes teaches **intrapericardial** injection rather than **intraperitoneal** injection. None of the cited references teach the claimed method of the instant invention and the reporter genes and other genes used in the cited reference are not the alpha-MHC gene as claimed. Since the unpredictable nature of the in vivo gene therapy in general and specifically the in vivo myocardial gene therapy as discussed above, each in vivo gene therapy has to be considered individually. One successful in vivo gene therapy cannot be extrapolated into success for another gene therapy in vivo. Further, the genes used in the cited references are different from the alpha-MHC gene used in the instant invention. It was well known in the art that different proteins have different biological functions. As discussed above, numerous factors complicate in vivo gene therapy with respect to predictably achieving levels and duration of gene expression. These factors include the gene used, the level of mRNA produced, the stability of the mRNA

produced, the amount and stability of the protein produced, the biological function of the protein, and the disease being treated. Therefore, whether administration of a transgene encoding alpha-MHC to a human would be able to provide therapeutic effect for treating myocardial failure in vivo would require specific guidance and evidence, which is absent in the instant invention. Thus, the cited references could not constitute a success of the myocardial gene therapy in vivo as claimed.

Appellants cite references Lin et al., 1990, Stradford-Perricaudet et al., 1992, Von Harsdorf et al., 1993, French, 1994, Lee et al., 1996, Doffin et al., 1996, and Kypson et al., 1998, and argue that those references teach direct injection of genes into the myocardium (brief, p. 7, lines 2-7). This is not found persuasive because of the reasons of record and the reasons set forth above regarding the cited references discussed above. Furthermore, the cited references only teach direct injection of genes into the myocardium, however, the claims encompass delivering the transgene encoding alpha-MHC via various administration routes, for example, oral administration, intramuscular administration, intravenous administration, intraperitoneal administration, and subcutaneous administration etc. As discussed above, numerous factors complicate in vivo gene therapy with respect to predictably achieving levels and duration of gene expression which have not been shown to be overcome by routine experimentation. These factors include, the fate of the DNA vector itself (volume of distribution, rate of clearance into the tissues, etc.), the *in vivo* consequences of altered gene expression and protein function, the fraction of vector taken up by the target cell population, the trafficking of the genetic material within cellular organelles, the rate of degradation of the DNA, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, and the

protein's compartmentalization within the cell, or its secretory fate, once produced. These factors differ dramatically based on the vector used, the protein being produced; and the disease being treated. The specification fails to provide adequate guidance and enabling disclosure for the full scope of the invention claimed. Whether administration of a transgene encoding alpha-MHC to a human via various administration routes would be able to provide therapeutic effect for treating myocardial failure in vivo would require specific guidance and evidence, which is absent in the instant invention. Thus, the cited references could not constitute a success of the myocardial gene therapy in vivo as claimed.

Appellants argue that reference Fromes proved that intra-pericardial injection leads to an efficient and safe strategy to deliver a transgene to the heart and Hajjar used a catheter-based technique to successfully alter cardiac function in rat hearts (brief, p. 7, lines 7-15). This is not found persuasive because of the reasons of record and the reasons set forth above regarding the cited references discussed above.

Appellants argue that reference Schroder et al., 2000, teaches addition of anti-CD4 monoclonal antibodies improved gene transfer into rat cardiac grafts, O'Donnell et al., 2001, showed that sarcoplasmic reticulum ATPase (SERCA) could be expressed in cardiac myocytes, del Monte et al., 2001, showed effective transfer of and expression of SERCA2a into rat heart, and Li et al., 2003, showed that an AAV vector could be used to transfer a reporter gene and a therapeutic gene into the heart of a hamster, and Yue et al., 2003, treated a cardiovascular disease using an AAV vector expressing microdystrophin and showed improvement of cardiovascular function. This is not found persuasive because of the reasons of record and the reasons set forth above regarding the cited references discussed above.

Appellants cite reference Poller et al., 2003, and argue that Poller states "the important goal of long-term stability of therapeutic vectors has recently been achieved in animal models using vectors derived from adeno-associated viruses" (brief, p. 8, lines 6-10). This is not found persuasive because of the reasons of record and the reasons set forth above. Reference Poller only refers to AAV vector, however, the claims encompass delivering the transgene encoding alpha-MHC by using various vectors, such as plasmid, adenoviral vector, retroviral vector, Herpes Simplex Virus vector, and liposome etc. As discussed above, a resolution to vector targeting of gene therapy in vivo has not been achieved in the art, and the design hurdles for all vectors are the need to increase the efficiency of gene transfer, to increase target specificity and to enable the transferred gene to be regulated. Numerous factors (discussed above) complicate in vivo gene therapy with respect to predictably achieving levels and duration of gene expression. These factors differ dramatically based on the vector used, the protein being produced, and the disease being treated. The specification fails to provide adequate guidance and enabling disclosure for the full scope of the invention claimed. Whether administration of various vector containing a transgene encoding alpha-MHC to a human via various administration routes would be able to provide therapeutic effect for treating myocardial failure in vivo would require specific guidance and evidence, which is absent in the instant invention. Thus, the cited reference could not constitute a success of the myocardial gene therapy in vivo as claimed.

Appellants argue that an applicant need not have actually reduced the invention to practice prior filing, appellant need not describe all actual embodiment, and nowhere in the patent law stated that the use of post-filing references is prohibited from enabling a claimed method. Appellant further argue that the claimed method is generic to treating the disease and

the methods disclosed by the cited references all fall within the body of art claimed by the current invention (brief, p. 8-9). This is not found persuasive because of the reasons of record and the reasons set forth above. Indeed, one does not need to describe all actual embodiments, however, appellants must provide sufficient enabling disclosure for the full scope of the invention claimed but fail to do so in the instant invention. A post-filing reference can be used to enable a claimed method as long as the method practiced in said reference is the same as the claimed method and an enabling disclosure is disclosed in said reference. However, the claimed method of the instant invention is drawn to a method of treating myocardial failure in a human by administering a transgene encoding alpha-MHC and expression of alpha-MHC provides improvement in left ventricular ejection fraction. The claimed method is not generic to treat any disease, rather, it specifically reads on treating myocardial failure with a transgene encoding alpha-MHC in a human. The claimed method is totally different from the gene therapy method taught in the cited references by appellants. None of the cited references teach the claimed method of the instant invention and the reporter genes and other genes used in the cited reference are not the alpha-MHC gene as claimed. Since the unpredictable nature of the in vivo gene therapy in general and specifically the in vivo myocardial gene therapy as discussed above, each in vivo gene therapy has to be considered individually. For the reasons discussed previously, one successful in vivo gene therapy cannot be extrapolated into success for another gene therapy in vivo. Further, the genes used in the cited references are different from the alpha-MHC gene used in the instant invention. It was well known in the art that different proteins have different biological functions. As discussed above, numerous factors complicate in vivo gene therapy with respect to predictably achieving levels and duration of gene expression. These factors

include the gene used, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, the biological function of the protein, and the disease being treated. Therefore, whether administration of a transgene encoding alpha-MHC to a human would be able to provide therapeutic effect for treating myocardial failure in vivo would require specific guidance and evidence, which is absent in the instant invention. Thus, the cited references by appellants fail to provide enabling disclosure for the claimed method of the instant invention for the reasons discussed above.

(11) Related Proceeding(s) Appendix

No decision rendered by a court or the Board is identified by the examiner in the Related Appeals and Interferences section of this examiner's answer.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

Shin-Lin Chen

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